

## CASE REPORTS

### AORTIC EMBOLISM DUE TO MYXOID TUMOUR ASSOCIATED WITH MYOCARDIAL CALCIFICATION

BY

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Although about 200 cardiac myxomata have been reported (Landing and Farber, 1956), the controversy surrounding their exact nature, whether neoplastic or thrombotic, justifies the recording of any new association. In the present case a calcified myxoma was intimately related to an extensive and encircling calcification of the mitral annulus. Some calcification of the mitral annulus is fairly common in old age. Geill (1950) pointed out that it was not related to preceding endocarditis. A significantly higher incidence in Paget's disease of bone than in control series was found by Harrison and Lennox (1948). On the other hand, calcification in cardiac myxomata is very rare. Lekisch (1957) described a left atrial myxoma with calcification and cartilage formation in its base but, as with the majority of myxomata, there was no evidence of other myocardial disease.

#### *Case History*

W. K., a man aged 57, who had previously been in excellent health, was walking to the station at 8 a.m. on December 29, 1960, when he was suddenly halted by pain extending from both feet up the legs to the lower abdomen. Within two minutes both legs became completely numb and paralysed and he collapsed. He also complained of mild low backache. There was no previous history of angina or other cardiac symptoms. On admission to hospital the blood pressure was 130/80 mm. Hg. The heart sounds were normal with no added sounds. Both lower limbs were cold, pulseless, and paralysed with loss of sensation. A provisional diagnosis of dissecting aneurysm of the abdominal aorta or of an aortic saddle embolism was made, and the patient was transferred to the surgical unit at St. Mary's Hospital, where the findings on admission were as follows.

The pulse rate was 86 a minute with a regular rhythm. The apex beat was not palpable. The heart sounds were normal and no murmurs were heard. The blood pressure was 120/80 mm. Hg. The lower limbs were pale and pulseless. The respiration rate was 30 a minute. No other abnormality was detected. The abdomen was considerably distended and bowel sounds were absent. No aortic swelling was palpable, and no bruit was audible over the aorta. Examination of the central nervous system revealed complete sensory loss, flaccid paralysis, and loss of reflexes in the lower limbs.

After admission the patient's general condition further deteriorated, and the systolic blood pressure fell to 60 mm. Hg. Resuscitative measures, including blood and plasma transfusion, were commenced. Catheterization of the bladder yielded 50 ml. of clear urine. At 6 p.m., 10 hours after his initial collapse, the patient was taken to the theatre, but in the anaesthetic room his general condition worsened and cardiac arrest occurred. Thoracotomy was immediately performed and after ten minutes' cardiac massage the heart beat was restarted and ventricular fibrillation was converted to normal rhythm by means of an electrical defibrillator.

The abdomen was then opened through a left paramedian incision (J.R.K.). The entire intestine was grossly distended, the large bowel was a light purplish colour and the greater part of the small bowel was similarly affected. A small quantity of malodorous free fluid was present in the peritoneal cavity. There

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was no pulsation in the superior or inferior mesenteric arteries. The abdominal aorta felt extremely hard and solid in its upper part. Before further exploration of the aorta was possible ventricular fibrillation again occurred. This proved to be irreversible.

*Necropsy Findings.* This was an obese middle-aged man with a recent left paramedian surgical incision and a left thoracotomy wound extending along the fifth left costal interspace.

*Cardiovascular System.* About 20 ml. of blood were present in the pericardial sac and there were several petechiæ in the epicardial fat, findings that could be explained by the recent cardiac massage. The heart (weight 520 g.) was enlarged due to hypertrophy and some dilatation of the left ventricle. The dilated left atrium showed slight and diffuse endocardial thickening. The mitral valve (circumference 11 cm.) had minimal fibrous adhesion of several of the chordæ tendineæ. Calcification of the mitral annulus extended down in nodular fashion into the posterior wall of the ventricle, where it projected slightly on both endocardial and epicardial surfaces and upwards into the posterior wall of the left atrium. Here the calcification replaced the entire thickness of the muscle and appeared as an eroded area, 2.5 cm. in diameter, just above the posterior mitral cusp (Fig. 1). The extent of the calcification is seen in the radiogram of the specimen (Fig. 2). The heart showed no other gross abnormality and the other valves and the coronary arteries were normal.

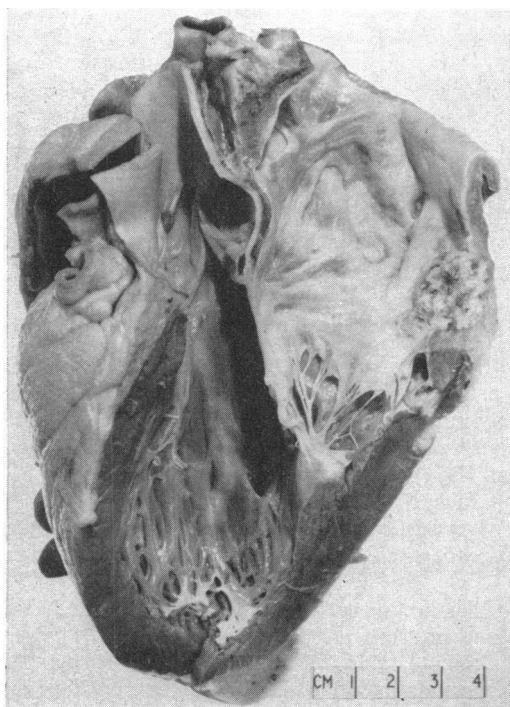


FIG. 1.—Anterior view of dissected left ventricle and atrium, showing the eroded calcified base of the myxoma on the extreme right of the photograph and the calcification in the ventricular wall below the mitral annulus.



FIG. 2.—Radiogram of heart and aortic embolus placed at bottom right-hand corner. Note the extensive calcification extending far beyond the base of the myxoma in the left atrium.

The aorta was mildly atheromatous, and just below the diaphragm and down to the level of the renal arteries it was completely occluded by an ovoid embolus measuring  $3 \times 2 \times 2$  cm. This structure was smooth except at its upper end, where it had an irregular calcified surface which matched exactly the eroded area on the posterior wall of the left atrium (Fig. 2). Reconstruction also showed that the smooth polypoid end of the embolus could be fitted into the dilated atrial appendage. The branches of the aorta were normal.

On sectioning, the embolus was seen to be irregularly calcified, most heavily towards its base, and myxomatous. Histologically it consisted largely of fibrous tissue, hyaline in some areas and in others

distinctly myxomatous with relatively few nuclei (Fig. 3). Extensive amorphous calcification was present and this was greatest at its base. Some areas were rich in elastic and collagenous fibres which in places outlined old vascular channels. Similar appearances were found in the heart at the site of the origin of the embolus. The amorphous calcification, with bone formation in places, extended into both atrial and ventricular myocardium and was surrounded by hyaline fibrous tissue. Wherever the calcification abutted onto the endocardium, and only there, some proliferation of fibrous tissue with increase in mucinous ground substance had occurred (Fig. 4). This material stained positive with mucicarmine stain and the periodic acid Schiff reaction, and staining with toluidine blue showed metachromasia. Minute amounts of hæmosiderin were seen in the stroma.



FIG. 3.—Myxomatous area. (H. and E.  $\times 245$ )

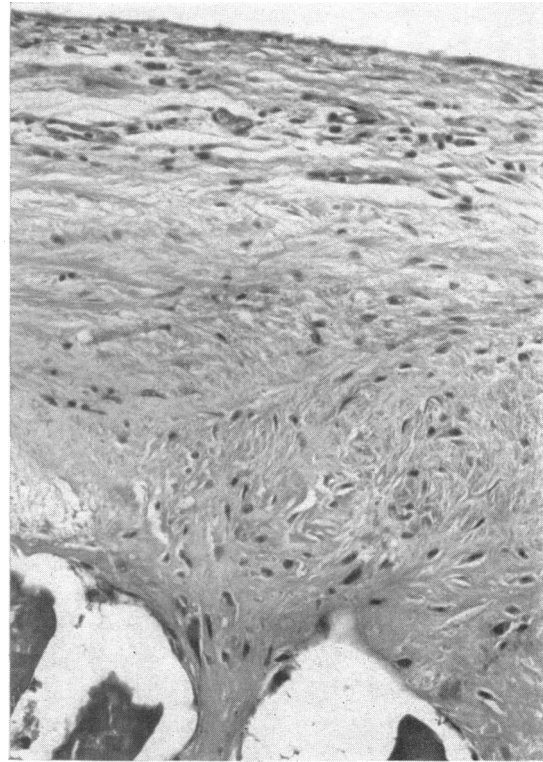


FIG. 4.—Endocardial reaction with mucinous ground substance over ventricular calcification. (H. and E.  $\times 225$ .)

*Alimentary System.* A purplish discoloration affected the lower two-thirds of jejunum, ileum, and proximal half of the colon. As the congested bowel showed moderate post-mortem autolysis on microscopy, early necrotic changes could not be detected with certainty. The liver showed passive congestion.

*Other Systems.* There were no features of note, apart from congestion and œdema of the lungs with basal collapse. Autolytic changes in the kidneys obscured evidence of necrosis.

#### Discussion

This cardiac lesion was completely silent, even after the fracture of the myxoma at its base and its launching into the left side of the heart and subsequent impaction in the upper abdominal aorta. Obstruction at this level is rare. If circumstances had permitted embolectomy, recovery might have been expected. Several successful resections of cardiac myxomata have been reported (Scannell, Brewster, and Bland, 1956) and occasional aortic embolism due to large fragments of myxoma or to

thrombus which had formed in the heart secondary to the myxoma has also been recorded (Carter, Lowe, and Hill, 1960; Edwards and Johnson, 1959; Brewin, 1951). In the successful aortic embolectomy performed in one of Carter's cases by Professor D. M. Douglas the patient was well eight months after operation and later angiocardiology failed to reveal an intra-atrial myxoma. It is not known how often intracardiac surgery will prove necessary after successful embolectomies of myxomata. In the present case a similar thrombotic or reactive process would probably have recurred on the eroded calcified base in the left atrium. In this respect the basic pathology was the calcification in the mitral annulus with its nodular extensions into surrounding myocardium far beyond the base of the polypoid tumour. It may be significant that this reaction to amorphous calcification was relatively acellular and non-mucoid except on the endocardial aspects. The site of origin of the tumour appears to have been determined by the proximity of a large nodule of calcification to the endocardium, and it is difficult to believe that, initially at any rate, this was other than a reactive process or mural thrombosis followed by organization. The endocardium over the ventricular calcification was already showing early reactive changes similar to those in the tumour. Cases previously described have not shown such a clear-cut relation to a pathological process in the myocardium.

#### Summary

A case of aortic embolism due to detachment of a left atrial myxoma is described. The relation of the myxoma to underlying calcification and ossification of the mitral annulus and adjacent myocardium suggests that the myxomatous reaction was secondary to the extensive and preceding myocardial calcification and that this was the more basic pathological change.

We are indebted to Mr. J. R. Kenyon for permission to record the clinical findings and for his helpful criticism and to Dr. I. Milne, H.M. Coroner for North London, for allowing publication of the necropsy findings.

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